



## Improving the Effectiveness of Treatment of Muscle Hypotonia in Children

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**Abstract:** The purpose of the research. To develop proposals and recommendations for early diagnosis of muscle hypotonia syndrome in children, development of criteria for determining the level of severity, and determination of treatment measures, improving electrophysiological (ENMG, SEP) diagnostic methods.

In the period of 2022-2023, 110 children of early age (0-3) with muscle hypotonia syndrome were comprehensively examined in the department of "Childhood Nervous Diseases" of the 1st Children's Clinical Hospital of Tashkent City. In our research, we divided the patients with muscle hypotonia syndrome, that is, the representatives of each group (group 1,2,3,4) into 2 groups.

Representatives of the 1st group (47 people): traditional medical treatments, i.e. medical treatment + rehabilitation. Medicinal treatment includes nootropic, blood circulation improving, acetylcholinesterase drugs and group B vitamins. Representatives of the 2nd group (63 people): rPMS + rehabilitation. Treatment procedures were continued every month for 10 days from 3 to 6 months. Patients underwent ENMG and SSChP examination before and after treatment. , in our patients who received rPMS + rehabilitation, ENMG indicators were significantly higher than in patients who received conventional treatment.

In conclusion, rPMS+ rehabilitation resulted in a significant and short-term reduction in the latency period of SSChP peaks, an increase in the conduction of nerve impulses from the somatosensory pathway in response to n. medianus stimulation.

Even when rPMS was applied peripherally, significant efficacy was achieved in improving somatosensory cortical conductance in the SSChP study of patients with

central MGS. Our results showed that since rPMS is the hub of spinal cord descending, ascending and segmental nerve signals, non-invasive rPMS has the ability to simultaneously change cortical, corticospinal, spinal cord motor activity and conductance and excitability in peripheral nerves.

**The purpose of the research.** To develop proposals and recommendations for early diagnosis of muscle hypotonia syndrome in children, development of criteria for determining the level of severity, and determination of treatment measures, improving electrophysiological (EMG, ENMG, ChP) diagnostic methods.

**Material and test methods.** During 2022-2023, 110 children with muscle hypotonia syndrome at an early age (0-3) were comprehensively treated in the Department of "Childhood Nervous Diseases" of Tashkent City 1st Children's Clinical Hospital and on the basis of a scientific contract with "Neurocell and Co." was examined in a private clinic. 57 (51.8%) boys and 53 (48.2%) girls participated in the examination. All children were carefully examined clinically and neurologically according to the generally accepted method. The examined patients were divided into 4 groups according to the results of clinical and anamnestic, ultrasound, dopplerographic and electrophysiological examination.

Children with muscle hypotonia of central type (group 1) had 32 (29), children with MGS of peripheral type (group 2) had 25 (23), children with mixed, that is, MGS with damage of both central and peripheral type (3 -group) accounted for 43 (39%) and chromosomal diseases with muscle hypotonia (group 4) accounted for 10 (9%).

In our research, we divided the patients with muscle hypotonia syndrome, that is, the representatives of each group (group 1,2,3,4) into 2 groups. Representatives of the 1st group (47 people): traditional medical treatments, i.e. medical treatment + rehabilitation. Medicinal treatment includes nootropic, blood circulation improving, acetylcholinesterase drugs and group B vitamins. Representatives of the 2nd group (63 people): rPMS + rehabilitation. All patients in this group underwent neurosonography and EEG before receiving rPMS treatment. This is because patients without brain tumor foci were selected for this group. The patient's parents were given detailed information about the procedure and their consent was obtained for the procedure. Treatment procedures were continued every month for 10 days from 3 to 6 months. Patients underwent ENMG and SSChP examination before and after treatment. The results were entered into the patient's medical history.

According to the results of pre-treatment ENMG examination of patients who received traditional medical treatment, the mean response amplitude of n.peroneus motor fiber M in group 1 patients was  $4.25 \pm 0.11$  (right)  $3.7 \pm 0.16$  (left)  $R < 0.01$ ,  $2.46 \pm 0.1$  (right)  $2.24 \pm 0.1$  (left)  $P < 0.01$  in group 2,  $2.58 \pm 0.1$  (right),  $2.62 \pm 0.12$  (left)  $P < 0.01$ ,  $2.6 \pm 0.21$  (right)  $3 \pm 0.47$  (left)  $P < 0.01$  in group 4,  $4.25 \pm 4.25$  in group 1 patients after treatment  $0.11$  (right)  $3.7 \pm 0.16$  (left)  $R < 0.01$ ,  $2.55 \pm 0.2$  (right)  $2.46 \pm 0.1$  (left)  $P < 0.01$ , in group 3  $2.61 \pm 0.1$  (right),  $2.91 \pm 0.12$  (left)  $P < 0.01$ , in group 4  $2.6 \pm 0.21$  (right)  $3 \pm 0.47$  (left) was  $P < 0.01$  (Figure 1).

By the 2nd method, that is, when rPMS+ rehabilitation was applied to the patients, according to the results of the ENMG examination before the treatment, the average indicator of the amplitude of the motor fiber of the n.peroneus M-response was  $3.26 \pm 0.11$  (right)  $3.12 \pm 0.16$  (left)  $R < 0.01$ , in group 2 patients  $1.99 \pm 0.1$  (right)  $2 \pm 0.1$  (left)  $P < 0.01$ , in group 3  $2.32 \pm 0.1$  (right),  $2.34 \pm 0.12$  (left)  $P < 0.01$ , in group 4 it was  $3.1 \pm 0.21$  (right)  $2.4 \pm 0.47$  (left)  $P < 0.01$ , after treatment 1 -group patients  $4.65 \pm 0.15$

(right)  $4.5 \pm 0.13$  (left)  $R < 0.01$ , 2nd group patients  $3.8 \pm 0.2$  (right)  $4.2 \pm 0.1$  (left)  $P < 0.01$ ,  $3.8 \pm 0.1$  in group 3 (right),  $4.15 \pm 0.12$  (left)  $P < 0.01$ ,  $3.72 \pm 0.12$  in group 4 (right) was  $3.59 \pm 1.1$  (left)  $P < 0.001$

According to the results of pre-treatment ENMG examination of patients who received traditional medical treatments, the mean index of M-response amplitude of n.tibialis motor fiber in group 1 was  $8.22 \pm 0.2$  (right side),  $9.3 \pm 0.17$  (left side)  $P < 0.01$ , in group 2  $6.5 \pm 0.2$  (right side),  $7.1 \pm 0.19$  (left side)  $P < 0.01$ , in group 3  $9 \pm 0.17$  (right side),  $8.9 \pm 0.21$  (left side)  $P < 0.01$ , in group 4  $6.64 \pm 0.5$  (right side),  $7.6 \pm 0.29$  (left side)  $P < 0.01$ , after treatment in group 1  $8.5 \pm 0.5$  (right side),  $9.5 \pm 0.2$  (left side)  $P < 0.01$ , in group 2  $6.58 \pm 0.1$  (right side),  $7.2 \pm 0.12$  (left side)  $P < 0.01$ , in group 3  $9 \pm 0.14$  (right side),  $8.9 \pm 0.15$  (left side)  $P < 0.01$ , in group 4 it was  $6.8 \pm 0.34$  (right side),  $7.8 \pm 0.12$  (left side)  $P < 0.01$

According to the ENMG examination results of our patients in group 2 before treatment, the average index of M-response amplitude of n.tibialis motor fiber in group 1 was  $8.9 \pm 0.2$  (right side),  $9.2 \pm 0.17$  (left side)  $P < 0.01$ , in group 2 were  $5.9 \pm 0.2$  (right side),  $6.2 \pm 0.19$  (left side)  $P < 0.01$ , in group 3 it was  $11.3 \pm 0.17$  (right side),  $11.6 \pm 0.21$  (left side)  $P < 0.01$ ,  $7 \pm 0.5$  (right side),  $7.3 \pm 0.29$  (left side)  $P < 0.01$  in group 4 if he did, after treatment in group 1 it was  $11 \pm 0.5$  (right side),  $12.3 \pm 0.2$  (left side)  $P < 0.01$ , in group 2 it was  $10.6 \pm 0.1$  (right side),  $10.2 \pm 0.12$  (left side)  $P < 0.01$ , in group 3 it was  $11.3 \pm 0.14$  (right side),  $11.6 \pm 0.15$  (left side)  $P < 0.01$ , in group 4 it was  $10.8 \pm 0.34$  (right side),  $10.2 \pm 0.12$  (left side)  $P < 0.01$  (Fig. 4). All patients underwent post-treatment SEP and compared with pre-treatment SEP results.

**Table 1. Mean pre-treatment SEP score of patients receiving conventional medical treatments.**

Guruhlar	N9	N13	N20	P25	N9-N13	N9-N20	N13-N20
1-guruh	8,66	12,5	24,6	28,6	3,84	15,9	12
2-guruh	8,88	16,2	19,3	25,05	7,32	10,42	3,1
3-guruh	8,85	16,1	24,8	28,8	7,25	15,95	8,7
4-guruh	7,3	13,9	24	28,8	6,6	16,7	10,01

According to the results of the scientific research, SEP examination of the patients who received traditional medical treatments before the medical treatments, the latent period of the N13, N20 and P25 peak in group 1 was on average  $12.5 \pm 0.2$  ms (N13) and  $24.6 \pm 0.5$  ms (N20)  $28.6$  ms (P25), the N9-N13 interpeak interval was  $3.84 \pm 1.6$  ms, the N9-N20 interpeak interval was  $15.9 \pm 0.1$  ms, and the N13-N20 interpeak interval was  $12 \pm 1.2$  ms ( $P < 0.01$ ), after treatment, the peak latency of N13 was  $12.5 \pm 1.1$ , the latency of N20 was  $24.3 \pm 0.4$  ms, and the latency of P25 was  $28.2 \pm 0.4$  ms, N9-N13, N9-N20 and N13-N20 peak intervals showed  $3.6 \pm 0.3$  ms,  $15.4 \pm 0.3$  ms and  $11.8 \pm 0.2$  ms respectively Table 3.

**Table 2. Mean post-treatment SEP examination of patients who received conventional medical treatments.**

	N9	N13	N20	P25	N9-N13	N9-N20	N13-N20
1-guruh	8,9	12,5	24,3	28,2	3,6	15,4	11,8
2-guruh	8,9	16	19,3	25,05	7,1	10,4	3,3
3-guruh	8,8	15,71	24,5	28,5	6,9	15,7	8,8
4-guruh	9,25	14,7	23,8	28,45	5,45	14,5	9,1

SEP examination of patients who received traditional medical treatment before treatment showed that the latency period of N13, N20 and P25 peak was on average  $16 \pm 0.2$  ms (N13) and  $19.3 \pm 0.5$  ms (N20)  $25.05 \pm 0.2$  ms (P25), N9-N13 inter-peak interval  $7.1 \pm 1.6$  ms, N9-N20 inter-peak interval  $10.4 \pm 0.1$  ms, N13-N20 inter-peak interval  $3.3 \pm 1.2$  ms ( $P < 0.01$ ), after treatment, the latency period of N13 peak

was  $12.78 \pm 0.1$ , the latency period of N20 peak was  $18.95 \pm 0.4$  ms, the latency period of P25 peak was  $25.1 \pm 0.4$  ms, N9-N13, N9-N20 and N13-N20 peak intervals showed  $8.27 \pm 0.3$  ms,  $10.45 \pm 0.3$  ms and  $2.18 \pm 0.2$  ms, respectively.

In group 3, the latency period of N13, N20 and P25 peak was on average  $15.7 \pm 0.2$  ms (N13) and  $24.1 \pm 0.5$  ms (N20),  $28.5 \pm 0.2$  ms (P25). , the N9-N13 interpeak interval was  $6.9 \pm 1.6$  ms, the N9-N20 interpeak interval was  $15.7 \pm 0.1$  ms, and the N13-N20 interpeak interval was  $8.8 \pm 1.2$  ms ( $P < 0.01$ ), after treatment the latency period of N13 peak was  $16.4 \pm 0.1$ , the latency period of N20 peak was  $25.3 \pm 0.4$  ms, the latency period of P25 peak was  $28.5 \pm 0.4$  ms, N9 -N13, N9-N20 and N13-N20 interpeak interval showed  $7.8 \pm 0.3$  ms,  $16.7 \pm 0.3$  ms and  $8.9 \pm 0.2$  ms respectively. In group 4, before conventional treatment, the following indicators were respectively  $8.5 \pm 0.4$  ms (N13)  $13.36 \pm 0.4$  ms (N20)  $24.3 \pm 0.4$  ms (P25),  $5.45$  ms ( N9-N13 ), showed  $14.5$  ms (N9-N20),  $9.1$  ms (N13-N20), after treatment, the peak latency of N13 was  $13.4 \pm 0.1$ , the latency of N20 peak was  $24.4 \pm 0.4$  ms, P25 peak latency was  $27.9 \pm 0.4$  ms, N9-N13, N9-N20 and N13-N20 inter-peak intervals are  $4.86 \pm 0.3$  ms,  $15.8 \pm 0.5$  ms,  $10.94 \pm 0.3$  ms.

**3- Table. Mean pretreatment SEP score of patients receiving rPMS+ rehabilitation treatments.**

Guruhlar	N9	N13	N20	P25	N9-N13	N9-N20	N13-N20
1-guruh	8,5	12,78	24,9	28,9	4,28	16,4	12,12
2-guruh	8,5	16,77	18,95	25,1	8,27	10,45	2,18
3-guruh	8,6	16,4	25,3	28,5	7,8	16,7	8,9
4-guruh	8,5	13,36	24,3	27,9	4,86	15,8	10,94

The mean pre-treatment SEP test of patients who received rPMS+ rehabilitation treatments, the latency period of N13, N20 and P25 peak in group 1 was on average  $12.78 \pm 0.2$  ms (N13) and  $24.9 \pm 0.5$  ms (N20)  $28.9 \pm 0.2$  ms (P25), N9-N13 inter-peak interval  $4.28 \pm 0.6$  ms, N9-N20 inter-peak interval  $16.4 \pm 0.1$  ms, N13-N20 inter-peak interval  $12.12 \pm 1.2$  ms ( $P < 0.01$ ), after treatment, the latency period of N13 peak was  $12.6 \pm 0.1$ , the latency period of N20 peak was  $18.95 \pm 0.4$  ms, and the latency period of P25 peak was  $25.25 \pm 0.4$  ms, N9-N13, N9-N20 and N13-N20 peak intervals showed  $3.54 \pm 0.3$  ms,  $10 \pm 0.3$  ms and  $6.4 \pm 0.2$  ms, respectively.

In group 2, which received rPMS+ rehabilitation treatments, the latency period of N13, N20 and P25 peak before treatment was on average  $16.7 \pm 0.2$  ms (N13) and  $18.95 \pm 0.5$  ms (N20),  $28.5 \pm 0.2$  ms (P25), the N9-N13 inter-peak interval was  $4.28 \pm 1.6$  ms, the N9-N20 inter-peak interval is  $16.4 \pm 0.1$  ms, and the N13-N20 inter-peak interval was  $12.12 \pm 1.2$  ms ( $P < 0.01$ ), after treatment, the peak latency of N13 was  $12.9 \pm 0.1$ , the latency of N20 was  $19 \pm 0.4$  ms, and the latency of P25 was  $25.25 \pm 0.4$  ms, N9-N13, N9-N20 and N13-N20 peak intervals showed  $3.8 \pm 0.3$  ms,  $9.9 \pm 0.3$  ms and  $6.1 \pm 0.2$  ms, respectively. In group 3, before the rPMS+ rehabilitation treatment, the following parameters were respectively  $16.4 \pm 0.4$  ms (N13),  $25.3 \pm 0.4$  ms (N20),  $28.5 \pm 0.4$  ms (P25),  $7.8$  ms (N9-N13),  $16.7$  ms (N9-N20),  $8.9$  ms (N13-N20). Table 4.

**Table 4. Mean post-treatment SEP score of patients who received rPMS+ rehabilitation treatments.**

	N9	N13	N20	P25	N9-N13	N9-N20	N13-N20
1-guruh	9,06	12,6	19	25,25	3,54	10	6,4
2-guruh	9,1	12,9	19	25,2	3,8	9,9	6,1
3-guruh	8,9	13	19	24,9	4,1	10,01	6
4-guruh	8,7	12,7	19,1	25,2	4	10,04	6,4

As a result of pre-treatment SEP examination in group 4 who received rPMS+ rehabilitation treatment, N13 peak latency was  $13.36 \pm 1.1$  ms, N20 peak latency was  $24.3 \pm 1.1$ , P25 peak latency was  $27.9 \pm 0.4$  ms, N9-N13, N9-N20 and N13-N20 inter-peak intervals were  $4.86 \pm 0.3$  ms,  $15.8 \pm 0.2$  and  $10.94 \pm 0.2$  ms, after treatment N13 peak latency is  $12.7 \pm 1.1$  ms, N20, P25 peak latency is  $19.1 \pm 0.52$  ms,  $25.2 \pm 0.4$  ms, respectively, N9-N13, N9-N20 and N13 -N20 interpeak interval was  $4 \pm 0.3$  ms and  $10.04 \pm 0.2$  ms,  $6.4 \pm 0.35$  ms, respectively. Table 4.

In conclusion, our patients treated with rPMS + rehabilitation had significantly higher ENMG scores than those treated with conventional treatment.

rPMS+ rehabilitation resulted in a significant and short-term reduction of the latency period of SEP peaks, an increase in the conductivity of nerve impulses from the somatosensory pathway in response to median nerve stimulation.

Even when rPMS was applied peripherally, significant efficacy was achieved in improving somatosensory cortical conductance in the SEP study of patients with central MGS. Our results showed that since rPMS is the hub of spinal cord descending, ascending and segmental nerve signals, non-invasive rPMS has the ability to simultaneously change cortical, corticospinal, spinal cord motor activity and conductance and excitability in peripheral nerves.

